



October 2012

## Jehovah's Witnesses and Blood Transfusion

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Jehovah's Witnesses believe that the Bible (Genesis 9:4, Leviticus 17:10, and Acts 15:29) prohibits ingesting blood and that Christians should therefore not accept blood transfusions or donate or store their own blood for transfusion. Specifically, their beliefs include:

- Blood represents life and is sacred to God. After it has been removed from a creature, the only use of blood that God has authorized is for the atonement of sins. When a Christian abstains from blood, they are in effect expressing faith that only the shed blood of Jesus Christ can truly redeem them and save their life.
- Blood must not be eaten or transfused, even in the case of a medical emergency.
- Blood leaving the body of a human or animal must be disposed of, except for autologous blood transfusions considered part of a current therapy.

Watch Tower Society publications teach that the Witnesses' refusal of transfusions of whole blood or its four primary components—red cells, white cells, platelets and plasma—is a non-negotiable religious stand and that those who respect life as a gift from God do not try to sustain life by taking in blood, even in an emergency. The following medical procedures are prohibited:

- Transfusion of allogeneic whole blood, or of its constituents of red cells, white cells, platelets or plasma.
- Transfusions of pre-operative autologous blood.

Members of the religion who voluntarily accept a transfusion are regarded as having disassociated themselves from the religion by abandoning its doctrines and are subsequently shunned by members of the organization.

For procedures where there is no specific doctrinal prohibition, individuals are to obtain details from medical personnel and then make a personal decision. The following procedures are left to the decision of individual members:

- Blood donation strictly for purpose of further fractionation of red cells, white cells, platelets or plasma for either allogeneic or autologous transfusion
- Transfusions of autologous blood as part of a current therapy
- Hemodilution using a continuous circuit
- Intraoperative blood salvage
- Heart-Lung Machine
- Dialysis
- Epidural Blood Patch
- Plasmapheresis
- Platelet Gel
- Fractions from blood plasma:
  - Albumin
  - Globulins
  - Cryoprecipitate
  - Cryosupernatant
  - Clotting factor concentrates including Factor VII, VIII and IX
- Artificial blood substitutes

A review of the literature regarding untransfused Jehovah's Witnesses found that nearly all those who died due to anemia had hemoglobin concentrations below 5 g/dL (Transfusion 1994;34:396-401). Of course, this conclusion is dependent on the underlying health of the patient and conservative management decisions made by the treating physicians.

The Blood Conservation Team at Saint Luke's Health System has extensive experience in managing anemia in Jehovah's Witnesses. Consults can be arranged by calling 816-932-6183.

## Reference Range Changes

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On October 16, the reference range for carbon dioxide was changed to 20-30 from 22-30 mEq/L. The reference range for anion gap was changed to 5-17 from 3-15.

## Reticulocyte Hemoglobin for Monitoring Iron Deficiency

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In the last issue of the Clinical Laboratory Letter, we announced a change in the reference range for reticulocyte count. In October, a new parameter, reticulocyte hemoglobin, was added to the reticulocyte count report.

Reticulocytes are immature red cells that circulate in peripheral blood for only 1 to 2 days. Measurement of reticulocyte hemoglobin content provides an indication of the amount of iron immediately available for incorporation into erythrocyte hemoglobin. The amount of hemoglobin in reticulocytes also corresponds to the amount that will be present in mature red cells. Lower than normal hemoglobin content is an indication of inadequate iron supply relative to demand.

This parameter is particularly important in assessing the iron status of patients receiving recombinant human erythropoietin (r-HuEPO), in whom the cellular requirements for iron are increased. Functional iron deficiency can hamper the effectiveness of r-HuEPO therapy.

The Kidney Disease Outcomes Quality Initiative (KDOQI) has included reticulocyte hemoglobin measurement in its guidelines for assessing the initial iron status of patients with chronic kidney disease on hemodialysis, as well as for monitoring IV iron and r-HuEPO replacement therapy. Studies have shown that reticulocyte hemoglobin <30 pg more accurately predicts functional iron deficiency than the combined use of ferritin <100 ng/mL and transferrin saturation <20%, particularly in patients on chronic hemodialysis who are receiving erythropoietin.

Reticulocyte hemoglobin is now reported with all reticulocyte counts. Reference range is 30 to 38 pg.

### **D-Lactic acidosis**

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A unique form of lactic acidosis can occur in patients with jejunioileal bypass or, less commonly, small bowel resection or other causes of the short bowel syndrome. Two major factors contribute to the overproduction of D-lactic acid in these situations: overgrowth of gram-positive anaerobes, such as Lactobacilli, and increased delivery of glucose and starch to the colon after the small bowel is bypassed, removed or diseased. Antimicrobial therapy can occasionally precipitate

D-lactic acidosis in susceptible individuals by promoting overgrowth of Lactobacilli.

D-lactic acid is absorbed into the systemic circulation and the ensuing acidemia tends to persist because D-lactate is not metabolized by lactate dehydrogenase, the enzyme that normally catalyzes the conversion of physiologically occurring L-lactate into pyruvate.

Patients with short bowel syndrome frequently demonstrate chronically elevated serum concentrations of D-lactate that are not sufficient to induce symptoms. In some patients, however, carbohydrate loading leads to severe and symptomatic D-lactic acidosis. Affected patients typically present with episodic metabolic acidosis, usually occurring after high carbohydrate meals, and characteristic neurologic abnormalities including confusion, cerebellar ataxia, slurred speech, and loss of memory. It is not known if these symptoms are actually due to D-lactate itself or to some other toxin produced in the colon and absorbed in parallel with D-lactate.

The diagnosis of D-lactic acidosis should be strongly considered in the patient presenting with an increased serum anion gap, normal serum concentration of L-lactate, negative plasma ketoacid level, negative urine ketones and the characteristic history, signs and symptoms.

The plasma anion gap may not be increased in proportion to the decrease in serum bicarbonate because D-lactate is not well reabsorbed by the renal tubules and is more readily excreted in the urine, thereby lowering the anion gap.

Confirmation of the diagnosis requires measurement of D-lactate that uses a special enzymatic assay employing D-lactate dehydrogenase. The standard assay for lactate uses L-lactate dehydrogenase and does not detect D-lactate. A random urine sample is the preferred specimen because D-lactate is readily excreted in urine. Reference range is 0.0-0.25 mmol/L. Increased levels are diagnostic of D-lactic acidosis.